Dear Editor,

I read with great interest the recent article by Mabilleau and Edmonds on the role of neuropathy on fracture healing in Charcot neuro-osteoarthropathy. There are, however, some inconsistencies that need to be addressed: on p 84, the authors state that “the aetiopathogenesis of diabetic Charco neuro-osteoarthropathy is still unclear”, while on p 87 they claim it begins with “trauma leading to intitial soft tissue and joint damage” resulting “in an exaggerated and uncontrolled inflammatory response which predisposes to multiple fractures and disruption of associated joints”, or with “bone resorption leading to intra-articular fractures and finally Charcot neuro-osteoarthropathy”. This assertion is implausible, as bone resorption and inflammation in Charcot neuro-osteoarthropathy invariably stop immediately upon cessation of any weight bearing activities (i.e. traumatisation) of the foot. The quoted changes in cytokine activities in acute Charcot neuro-osteoarthropathy, attributed to “bone resorption leading to ... fractures”, are the physiologic response to common fractures, most likely also in patients with neuropathy.

Sincerely,
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References